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THE ACCOMMODATION-CONVERGENCE RELATIONSHIP: A REFORMULATION*

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Ever since the publication of Donders' classic work on the eye, authorities seem to have accepted without much question the theory of the reflex nature of the accommodation-convergence relationship. It is true that Donders explicitly stated that within certain limits, these two functions are mutually independent so that for a given point of convergence there are what he called a positive and a negative range of accommodation. Nevertheless, although the reflex association can be broken up by lenses and prisms, artificially, it is generally accepted that normally the accommodation cannot be changed without effecting the convergence and vice versa. Two noteworthy exceptions to this traditional view are Worth and Stevens. Worth claims that it is the weakness of the fusion sense and not uncorrected hyperopia (as Donders claimed) that is the cause of convergent squint while.Stevens is practically alone in declaring unqualifiedly that the accommodation and the convergence are independent of each other both functionally and anatomically.[1a]

It seems to the writer that the latter attitude should be at present stressed if only as a salutory corrective against certain exaggerations that have followed the uncritical acceptance of what may be termed for the sake of convenience, the reflex theory. At any rate the mass of data that has accumulated since Donders' time makes a reconsideration and, possibly, a revaluation of the accommodation-convergence relationship desirable. In fact, certain observations have convinced the writer that the wisest, because the safest attitude, would be to regard the mutual independence of the two functions as the rule and to consider those cases in which the converse prevails as the exceptions. This would reverse the traditional attitude and radically affect both theoretical interpretations and clinical procedure. It is the purpose of this paper to establish a basis for this view by synthesizing all the evidence, most of it well known, that seem to support it.

(1) Before the histology of the oculomotor nucleus was known, it

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used to be taught that the accommodation and convergence are reflexly related because both the ciliary and the internal rectus are innervated by the same, third cranial nerve, and an impulse sent to one muscle simultaneously affected the other. The most recent investigations, however, have demonstrated the existence of separate cell-groups within the nucleus for specific innervations of individual muscles, although such localization cannot as yet be shown microscopically. The accumulation of embryological, anatomical, pathological and phylogenetical evidence in recent years has eliminated all doubt of the validity of this conception. The only question related to the problem that remains to be answered definitely is the identification of all the cell-groups in reference to the individual ocular muscles.

Specifically, there is little doubt that the sub-nucleus called the Edinger-Westphal group is the innervational center of the internal muscles, as established by experimental, clinical and post-mortem findings. Phylogenetically, it begins to appear before Perlia's nucleus, which latter is regarded as the innervation center for convergence.[2] Breuwer has shown the presence of the Edinger-Westphal group and the absence of Perlia's nucleus in animals like the Cetacea whose eyes are at the sides of the head. Such cases are living examples of accommodation without convergence. In the human embryo, also, the Edinger-Wesphal nucleus appears relatively early; it is not until the eyes swing around from the lateral position to the front of the head that Perlia's nucleus developes.[2]

Anatomically and physiologically, then, the suggested view of the accommodation and the convergence as separate functions is not without strong support. It is admitted that the fact of the location of their innervational centers in the same nucleus would presuppose the most delicate and flexible co-ordination between them. At the same time the separateness of the centers, physically, and the chronological differences of their development both in phylogeny and in ontogeny suggest strongly the possibility of mutually exclusive innervation.

(2) Pathologically, the evidence for this view is exceptionally strong. Thus, ophthalmoplegia may be external alone or internal alone; the individual may be able to exert his accommodation without his convergence or his convergence without his accommodation. In chronic ophthalmoplegia, it is the external ocular muscles that are generally affected while the ciliary and sphincter iridis are spared.[3] In an investigated case of external oph- In the human embryo, also, the Edinger-Westphal nucleus appears rela- but almost the entire remainder of the oculomotor nucleus was gone.[2]

(3) In the development of the human eyes after birth, the accommodation and convergence do not begin to operate synchronously. The former appears before the latter. Fixation of an object by each eye separately is automatic and instinctive, like the pupillary light reflex which is normally present at birth; but the cultivation of convergence and binocular vision requires education.[4a] Thus, the first five or six weeks after birth, the visual axes are not accurately converged when the infant fixes a near object, although the muscular balance for vertical movements seems, I

believe, normal. After this time, binocular vision begins to assert itself but is disturbed by the slightest influence. It is not until the age of five or six months that there is definite evidence of a desire for binocular vision, as shown by the ability of the infant to overcome a prism placed before one of the eyes, in which case the eye turns in the direction of the apex. It is possible, too, that it takes years before the child is educated to the higher type of adult binocular vision, and it has therefore been suggested that one cause of strabismus is the fact that reading and writing are taught too early in life before the practice of perfect binocular vision has been thoroughly mastered and maintained.[5]

We thus see that in the life of the individual, accommodation and convergence are developed separately in point of time and that while one faculty is instinctive and automatic, the other requires training and education for its development, the success of which depends upon individual mental capacity. When the habit-association of these functions becomes reflex in character, it is a conditioned and not an unconditioned reflex, a point which will be referred to later.

(4) The existence of uncorrected ametropia with single, binocular vision offers a well-known obstacle to the reflex theory. A hyperope or a myope may both exert the same degree of convergence at 13″ in order to secure binocular fixation on the maculae but the degree of accommodation is different in each case, both in reference to each other and in reference to the emmetrope who is looking at the same point.* In an attempt to solve this dilemma it has been suggested that each individual is born with his own particular accommodation-convergence relationship, the numerical value of which depends on his requirements but that this relationship is constant in the individual. The difficulty with this solution is that it applies only to congenital ametropia. In acquired ametropia, the individual starts out in life with a definite, habituated relationship that must change when hyperopia or myopia is acquired. These cases are difficult to explain on the basis of the reflex theory especially when the ametropia is high and binocular vision is unimpaired.

(5) Another well-known difficulty to this interpretation is offered by those cases in which there is myopia and esopheria or hyperopia and exophoria. These cases are by no means negligable in number. "In the experience of all, exophoria is found in a large group of cases in whom the refractive error is hyperopic."[6] Attempts to bring an explanation of these phenomena in line with the reflex theory sound suspiciously like rationalizations.

(6) The development of exophoria with the advent of presbyopia offers another difficulty. This phenomenon is often observed in practice. One authority[6] has attempted to explain it on the basis of a sclerosis of the nucleus resulting in a diminution of the powers of convergence and accommodation. It is well known, however, that normally the amplitude of convergence does not diminish with age, or else it diminishes very little.[7]

*In anisometropia, moreover, both eyes could not be converged to the same point if the accommodation-convergence relationship were reflex.

With or without exophoria, the increasing deficiency of accommodation with middle age while the convergence capacity remains the same. argues strongly for mutually autonomous functioning.

(7) The chief evidence in support of the reflex theory is the occurrence of myopia with exophoria and hyperopia with esophoria. The former association is commonly explained as being caused by the fact that the lessened innervation from the accommodation-centers (due to the myopia) produces a diminished innervation to the convergence-centers. Likewise, esophoria associated with uncorrected hyperopia is interpreted as a sympathetic over-stimulation of the convergence due to excess innervation of the ciliary. The obvious danger of such interpretations, however valid they may be in many cases, is the logical fallacy of *post quod Ergo Propter Quod*. The absence of a control in such investigations makes it impossible to state definitely whether or not the individual would have had the esophoria, say, without the hyperopia or vice versa. It is pertinent here to recall the criticism of Donders' theory of the hyperopic cause of convergent squint in so far as it was based on statistical evidence of relationship-occurrences. Thus, the frequency with which uncorrected hyperopia in children is associated with squint is not necessarily significant, for most children are hyperopic anyway and only a small percentage of them squint.[4b] Moreover, a statistical analysis of sample cases has shown that moderate degrees of hyperopia are more common than high degrees not only in those who squint but also in those who do not squint,[4c] and it is the moderate degrees that are supposed to cause the squint according to the reflex theory. Furthermore, the relative number of cases of hyperopia of more than 2.00 D. among the non-squinters was found to correspond closely with the number of this kind among the squinters. Another series of squint cases recorded in the order in which they appeared on the case-books, shows no definite relationship between the nature of the refractive error and the nature of the squint.[1b] Thus, in cases of exotropia, 23 were myopic, 38 hyperopic and 23 emmetropic; of esotropia, 18 were myopic, 20 hyperopic and 23 emmetropic. One authority found that in many cases of convergent squint and myopia, the best results were obtained by a full correction of the myopia,[4d] instead of an under-correction, as we would expect according to the reflex theory. Finally, the same authority discovered that elimination of overcorrecting "fogging" lenses that had been prescribed in cases of convergent squint, resulted in a lessening of the deviation in a few weeks.[4d]

It might also be pointed out that if the relationship between the accommodation and the convergence were fixed in most cases, there would be as many meter angles of heterophoria at definite distances as there are dioptres of ametropia. Actually, the amount of heterophoria associated with a given degree of ametropia varies widely among individuals.

One of the most convincing arguments in support of the reflex theory is the successful application of the rule that hyperopia should be fully corrected or overcorrected when associated with esophoria and under-

corrected when associated with exophoria; and that myopia should be fully corrected with exophoria and undercorrected with esophoria. Obviously, if we considered the accommodation and the convergence as independent of each other, we would correct the ametropia without reference to the muscular equilibrium, our only concern being the ciliary requirements. Now it is conceivable that the successful results that have followed the correction of the ametropia in accordance with the tonicity findings may have been due to the fact that the prescription happened to coincide with the demands of the ciliary. Thus, a full correction or even an overcorrection of hyperopia is often required whether or not it is associated with esphoria, especially in cases of spasm or hypertonicity of the ciliary.

It is significant that it is not the practice of refractionists to overcorrect the myopia even when there is a high degree of exophoria. This would seem to indicate that even the adherents of the reflex theory are careful to give the demands of the ciliary the prime consideration. This attitude seems to be justified by clinical experience which teaches that .25 D. of astigmatism is more liable to cause asthenopia in many cases than 3.00 prism D. of heterophoria.

(8) Even when the accommodation and convergence operate together for the fixing of a new object, their adjustments are not synchronously made. It is probable that convergence is effected first, followed a split-second later by accommodation, owing to the fact that it is more difficult to accommodate for a clear object seen double than to fuse two similar images which are blurred.[8]

(9) The relationship between the convergence and accommodation is not constant when the eyes are fixing objects that are not on the medial line. It can be shown geometrically that as the eyes turn to the right or the left as they fix an object, the degree of convergence relative to the accommodation becomes greater than in the median line up to a certain point.[9a] Beyond this point, the degree is less. As the eyes are continually fixing objects off the median plane for all distances, the postulation of a constant relationship between the accommodation and convergence can apply only to a relatively small part of the time that they are in use.

Aside from the geometrical considerations which make proportionate changes in the relationship necessary in order to avoid diplopia, the more the eyes look to either side, the greater is the physiological *difficulty* of converging, so that as far as effort is concerned, the excess of convergence over the accommodation increases with the lateral range.[9b] Thus, with accommodation at 10″ the degree of exophoria may be more than doubled as the eyes converge to a point 35 degrees to the right from the median line.

(10) Tscherning claims[10] that when an emmetrope fixes a near object and one of the eyes is occluded, it will continue to converge toward the point fixed by its mate "due to the relationship which exists between convergence and accommodation." Unfortunately for this theory, the eye under the conditions will *not* continue to converge but will turn out 3 or 4 degrees. This fact has been definitely established, especially by means of Maddox' "visual camera."[9c]

(11) Investigations into the behavior of both eyes when they are engaged in reading indicate that there are slight movements of the eyes during the fixation pauses and also that these pauses are more frequent than is needed to secure clear vision of the parts read, as shown by the fact that they remain the same in number when the print is removed to double the distance from the eyes. "This would seem," comments Prof. Fox, "to belittle our time-hallowed belief in the accommodative-convergence relationship."[11]

(12) Some individuals, especially artists, are known to have the power to contract their ciliary muscles at will without converging.[12]

(13) The influence of moderate degrees of heterophoria on the monocular and binocular amplitude of accommodation has been investigated. The results indicate that esophoria and exophoria have practically no influence,[13] which is contrary to what would expect on the basis of the reflex theory.*

(14) In their physiological state of rest, the eyes diverge slightly, a fact that is not revealed by the usual distant tonicity test because the test is conducted at a distance of only six meters. Yet the normal emmetrope has no equivalent "negative" accommodation. He sees clearly at a great distance with accommodation relaxed. Hence strictly speaking, slightly more convergence effort is needed for all distances than accommodative effort. But the discrepancy between them is even greater as the near point is approached and physiological exophoria (normally 3 to 6 prism D. at 13″) so-called is added to the distance exophoria. Thus even on the median line and in the case of the normal, young emmetrope, the relationship between the *efforts* of convergence and accommodation changes in value as the point of fixation moves from distance to near.

(15) It has been suggested that innervational impulses from the convergence-centers may be reflex from the retinae.[14] This is indicated by the fact that if prisms base-in are placed before the eyes, they will diverge, an act which most individuals cannot accomplish volitionally. Likewise, if a prism base-up or down is placed before one eye, it will rotate up or down while its mate maintains its level gaze. (In both these experiments we are assuming that the eyes are looking off into the distance so that the accommodative factor is eliminated.) Apparently it is the necessity for corresponding positions of the images and not the accommodative innervations that dictates the behavior of the rotators. It has also been suggested that the desire for single, binocular vision may be weakened by a blurring of the retinal images and increased when they are made clearer, with resultant modifications in the activity of the rotators. This theory helps to explain the decrease of exophoria when the hyperopia is fully corrected and similar phenomena that cannot be easily explained on the basis of the reflex theory.

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*For the amplitude of accommodation is supposed to be influenced by the degree and nature of the refractive error. See Lionel Laurance's "Visual Optics and Sight Testing," page. 89.

(16) If rotary prisms, base-in, are placed before the patient's eyes which are directed toward letters in the distance, and slowly increased in power, a point will often be reached when single binocular vision is still maintained but the letters start to blur. Apparently the continually increasing relaxation of the convergence has resulted in a sympathetic relaxation of the accommodation, as indicated by the fact that the blur can be cleared up by adding plus spheres before the eyes.

Now at first glance, this test appears to offer strong corroboration of the reflex theory. There are, however, two facts that militate against this view. In the first place, it has been noted that different individuals show different reactions to the test. Many scarcely notice the blur at all before diplopia commences; and when fusion is destroyed, some individuals see the two images clearly, others see them blurred.[15]

In the second place, it must be remembered that the conditions under which the reflex appears are highly artificial. In fact the phenomenon appears to fit neatly into the category of Pavlov's "artificial conditioned reflexes."[16a] By this method of "conditioning," Pavlov and his assistants managed to produce reflexes in the laboratory that did not exist in the natural life of the animal. It would be fallacious to infer that, because the accommodation-convergence reflex was produced under the conditions of violent dissociation of the two functions in the prism test, therefore the same reflex relationship obtains in the normal use of the eyes. On the contrary, Savage and Sheard, among others, have noted that "convergence cannot, except rarely, induce accommodation."[17a]

(17) Of all the objective tests, it is in the so-called dynamic skiametric test that the accommodation-convergence relationship has been of prime importance. As originally devised by A. J. Cross in the late eighties, the interpretation of the findings when lens-power is added before both eyes (that are fixed on the card attached to the retinascope) until the movement of the skiascopic shadow is neutral, is based on a 1:3 relationship between the accommodation and the convergence. The amount of accommodation locked with the required convergence at the distance tested, will not surrender to the lenses in the trial-frame; but any excess over this amount will be neutralized by the lenses. The excess or deficiency relative to this point represents the kind and degree of ametropia.

Sheard was among the first to announce the fallaciousness of this interpretation. Having discovered in all cases that slightly more convex and less concave power than is required, is found dynamically, he postulated a physiological *lag* of accommodation behind convergence[17b] to the extent of .50 D. to .75 D., which must therefore be subtracted from the dynamic findings to indicate the ametropia. Furthermore, there is evidence to indicate that the degree of the lag varies according to age and that it differs in the same individual at different near distances.[17c]

Nevertheless, in spite of this observed discrepancy, the reflex theory continues to dominate the literature on dynamic skiametry. The latest interpretation in this field, based on investigations of over 1,700 cases, depends entirely on the conception of the accommodation-centers and con-

vergence-centers as ends of a reflex arc, and therefore correlates the dynamic findings with the tonicity findings both for near and far.[18]

The dependability of the method of dynamic skiametry as far as convergence-accommodation relationships is concerned, is limited by the fact that the eye under observation is so dazzled by the light reflected from the retinoscopic mirror that it does not see the letters on the fixation-chart unless the illumination is considerably reduced.[20] As to the fact that the eye insists on accommodating to a certain degree in spite of the offered assistance of convex lenses, this is probably not due to the convergence, as the findings remain the same when the other eye is occluded. It may be simply a retinal reflex due to the automatic, habituated response of the delicately adjusted ciliary, a response that it is often difficult to repress entirely even with cycloplegics.[20]

(18) At least one case has been recorded where a diverging strabismus was changed to converging strabismus by the application of atropine. As this drug paralyzes the accommodation, the effect is opposite to that we would expect on the basis of the reflex theory.[1c]

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A consideration of the foregoing data leads to the almost inevitable conclusion that in the vast majority of cases, the accommodation and convergence are separate, autonomous functions. The requirements of single, clear binocular vision often demand changes in the proportion of their respective values, and this proportion at any given second will be determined by external and internal quantitative relationships which vary with the movements of the eyes. If the relationship between them were fixed or reflex, these changing requirements could not be met and serious impairment of visual comfort and efficiency would result. It is only by the co-ordination of separately innervated muscles—the degree of activity of each muscle being regulated by its individual need—that the marvelous adaptability of the eyes can be maintained. A reflex condition is a functional anomaly and should be regarded as exceptional, rather than as the rule.

On the other hand, it cannot be denied that such anomalies do exist. There are many cases of convergent squint associated with hyperopia where the eyes become straight as soon as the correcting lenses are placed before them, and turn in toward the nose as soon as they are removed. Nor can all of these cases be explained on the basis of a correction of the hyperphoria (which is regarded as the cause of the squint) by the action of the eyes in finding different levels in the lenses, hence creating a prism effect,—as one authority has claimed.[1d] The phenomenon often occurs even when the eyes are looking through the glasses at the same level and in cases where there is no hyperphoria. Furthermore, cases have been recorded where the patient would not accept the full correction in the postcycloplegic examination until prisms to correct the heterophoria were placed in the trial-frame.[19] It has also been observed that there is a difference between the tonicity findings recorded when the lens-correction is worn and when it is not worn in many cases. [It might be remarked that

in *some* of these cases, the discrepancy might have been due to the fact that the correcting lenses were not accurately centered with respect to the center of each pupil, a situation that is sometimes not attainable with the usual type of trial-frame (or nose-glasses) unless the lenses are of different sizes; to say nothing of the possibility that the optical center and the geometrical center of each lens did not coincide.]

The existence of this reflex relationship is explainable on the basis of the theory of conditioned reflexes. The individual requirements of the accommodation and convergence result in a habituated association in which they operate almost synchronously and frequently coincide in their quantitative effects. This is equivalent to the preliminary repeated and simultaneous stimulation of the skin and the offering of food (which caused the flowing of saliva) in Pavlov's experiments. When the skin was stimulated after a certain period, the saliva started to flow even though no food was offered. Similarly, in the reflex cases referred to, the continual excess innervation of the ciliary muscle results in excessive convergence. For the nearer object that would normally stimulate this degree of convergence is substituted the stimulus of excess accommodation and the same response is elicited.

The conditioned reflex is based on the phenomenon of irradiation of positive or negative stimulation in the motor area of the cortex.[16b] Thus, in cases of convergent squint caused by hyperopia, it is possible that strong stimulation of the accommodation-centers via the retinal reflex arc, irradiated into the immediately surrounding region of the cortex and thereby affected the convergence-centers. It has been found experimentally that one of the factors governing the extent of the irradiation is the intensity of stimulation of the primary center.[16c] This would explain why, in so-called reflex cases, convergent squint is more commonly associated with fairly high hyperopia than with slight hyperopia.

On the other hand, it has been demonstrated that the addition of a conditioned inhibitor can change a positive into a negative conditioned reflex.[16d] The degree of the inhibition depends in part on the intensity of the new stimulus.[16e] Thus, in a case of moderately high hyperopia in a child, the desire for single, binocular vision, if sufficiently strong, can inhibit the conditioned reflex of overconvergence. This suggests a possible reconciliation of Donders' and Worth's theories of the causes of squint in many cases: the fusion sense is too weak to act as an effective inhibitor of the positive conditioned reflex which causes the squint.

It is not, however, necessary to depend on the operation of inhibitors to explain why the accommodation-convergence relationship is not reflex in the majority of cases. The continually new adjustments in this relationship that are demanded by the exigencies of dynamic vision would seem to prevent the establishment of a reflex in the first place. In the early, impressionable years of life, the convergence-centers are trained not to fulfill a mathematical proportion with the accommodation but to obey the law of corresponding points of the images in the interests of fusion. This means that sometimes more, sometimes less, convergence is exerted

relative to the degree of accommodation, depending on the conditions of sight. The joining of the two functions quantitatively at any point is a coincidence, not a reflex. On the other hand, the refractionist should always be on guard to detect the exceptions to this rule.

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CHROMAGRAPHOLOGY

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Chapter VI.

Theoretical Cause and Diagnostic Value of Alterations

In considering now the theoretical causes and diagnostic value of the alterations which take place in the interpretation of colours by the human animal, we should like to make a statement which might be interpreted as sermonizing, but the reader must be assured that is not desired. It has always been easier to quit than to carry on, and as a consequence, we find in all the healing arts or professions those who find a solution to the above problem by saying: "There is no diagnostic significance in colour field interpretation." To us, this means that *Chromagraphology* is in the balance, therefor this chapter.

When one keeps in mind the comparatively few hours devoted in any of the medical courses given on the North American Continent, to the study of vision, it can be easily understood that the intricate problems regarding the *modus operandi* of the visual apparatus would be of too mysterious a nature to warrant understanding without a better background of basic knowledge involving refraction and allied subjects; yet, we find that the ophthalmologists of this country have realized a possibility in the study of colour interpretations for the reason that (as mentioned before) Drs. Ferree and Rand were commissioned by the Ophthalmologic Committee of the American Medical Association to go more deeply into this subject; and for that purpose, set aside apparatus, equipment, and personnel valued at over one-half million dollars.

It behooves us, then, in the profession of Optometry not to be reticent in our conceptions of the possibilities existing in this field; and it is a constant source of mild disappointment, (which fortunately acts as a stimulant to the writer), to find some of our lecturers and teachers making statements publicly and privately, which, in their true meaning, leave no allowance for the possibilities of colour field analysis, and yet those same statements are formed so shrewdly as to prevent criticism later for what bids fair to be a backward stand.

It has been the pleasure and privilege of the writer, during the forma-

tion of this work on *Chromagraphology,* to spend several weeks on the Pacific Coast observing carefully the work of leaders in that section. Men of the caliber of Dr. Thomas A. Brombach, Dr. Harry L. Fuog, Dr. Jesse Wolfe, to say nothing of many others whose names are not available at this moment, have built up very fine Optometric practices, and have been accorded recognition largely due to their belief in the usage and knowledge of diagnosis by colour field interpretations. This, of course, is the reason that the writer is encouraged to offset the doubts of those just mentioned, particularly in the east, believing that in time they will recognize the value and fall in line with some of our more progressive men in the west.

Probably one of the greatest reasons for our slowness in securing diagnostic information by way of colour fields is due to the fact that for a long time we looked at colour interpretations as purely physiological demonstrations; and we were inclined to base all of our reasoning on photochemical substance, its alterations and reactions. Then, too, some of the students of this work were inclined to base all of their determinations on intricate studies involving wave-lengths, nodal points, refractive indices, etc. As will be shown, these were not sufficient, and an end was soon had. Still later on, considerations were given to the physical defects as they were expressed in colour vision; and certainly writers presumed that colour studies were too much at variance, and set results could not be produced that would always be indicative of a particular disease. This again was insufficient.

It is only recently that the psychological aspect of colour vision has worked itself to the foreground; and this, in conjunction with it being considered as a factor in all of the branches of vision. It rightly should, for this something called "mind," which is only vaguely located, is the thing which keeps us all in a balance between sanity and insanity, therefore affecting the functions of our nervous system so dependent upon it, combined with the normal action of certain glands. It is reasonable, then, to expect that the fears, obsessions, complexes, and worries of our patients can and most certainly do modify vision. However, it is not the intention of the writer to focus, in a short-sighted manner, all considerations of colour field analysis on the psychologic factors, but rather to insist that we try to build our understanding in *Chromagraphology* by combining physiologic, physical, and psychological involvements.

As to the importance of colour field charting as an evidence in symptomatology to the optometrist, we find this of great value even if one disregards temporarily the splendid work of the western men just mentioned, together with Dr. William S. Farmer, Dr. Ephriam Friedman, and a host of others. For instance (pardoning a repetition, it being justified in this case as it is desired to strengthen the reader's belief), we find in "An Introduction to Clinical Perimetry," by H. M. Traquair, this statement:

> "In partial impairment of function, *colour vision is first diminished* and vision for white is demonstrably reduced only when the impairment has reached a certain degree."

In considering this statement, remember that Traquair speaks of many

of the perimetric signs found in glaucoma and other conditions as only confirmatory evidence, stating that when we find visual defects in the field for white, it simply serves to confirm the fact that glaucoma, for instance, is already an established condition. Reasoning back then—logically, for the optometrist should certainly be on the ever-watchful lookout for glaucoma, making sure that he does not pass such dangerous cases by as a part of purely refractive routine, he will be far safer by being able to establish failures in the visual field in colour because of its earlier incipiency.

Then, too, we can turn to the book—"Visual Field Studies," by R. I. Lloyd, and find on Page 40, this statement:

> "The Bjerrum sign is a large finger-like process from the blind spot, top or bottom, or from both places; reaching in the form of an arc, toward and often involving the fixing area. This is a late sign of glaucoma simplex, *is a colour defect at first,* does not often retreat if once established, and if it becomes a defect for white, there is little hope for improvement."

Here, then, is a further substantiation, for Dr. Lloyd speaks of the Bjerrum sign as being a late sign, which, if once established in the field for white furnishes little hope for success in the work of the ophthalmologist; but there is encouragement there for the optometrist when he says it is a colour defect at first, making this point very clear—that an earlier incipiency is available for the optometrist who studies his patient's colour fields.

Recently, while the writer was in Philadelphia, desiring a conference with Dr. George E. de Schweinitz, because of the fact that in 1896 he had written into his book, "Toxic Amblyopias," many of the statements that are now being made relative to colour field interpretations, a visit was made (at his direction) to the University Hospital in the Department of Ophthalmology, and observations made of the charting records at that institution. Correspondence following that observation brought forth the letter herewith which should be of further interest:

HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA
Philadelphia
Department of Ophthalmology

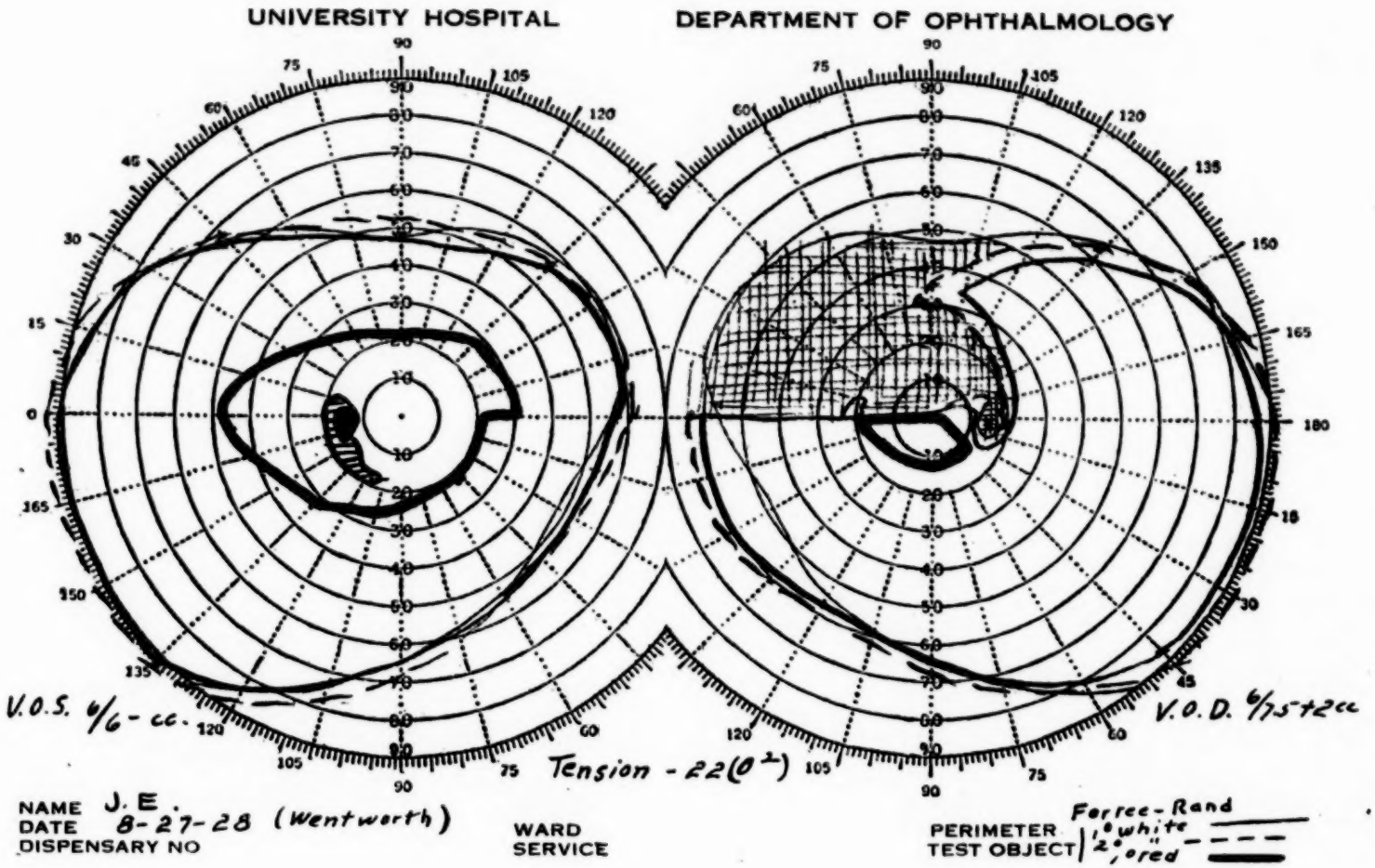
January 16, 1930.

"Dr. W. Jerome Heather,
666 W. Division St.,
Chicago, Illinois.

Dear Dr. Heather:

Thank you so much for the Engelking colours. They are most interesting. I deeply appreciate your kindness in obtaining them for me, and so promptly.

Yes, I have had several cases in which the Roenne step has been obtained in the red field and not in that for form, although it is never very large in such cases and generally has been found as the case improves under treatment rather than as a premonitory symptom. This may be due largely to the fact that I am most apt to see these cases for the first time after the condition has become severe. *I am enclosing a copy of the*


UNIVERSITY HOSPITAL
DEPARTMENT OF OPHTHALMOLOGY
V.O.S. 6/6 - cc.
V.O.D. 6/75+2cc
Tension - 22(0²)
NAME J. E.
DATE 8-27-28 (Wentworth)
DISPENSARY NO
WARD
SERVICE
PERIMETER Forree-Rand
TEST OBJECT 1° white
2° " red

fields in one case, however, in which it appears at the onset of glaucoma in the left eye; the disease having already gained considerable headway in the right eye as you can see from the chart. Note also the change in the colour blind spot as compared to that for form. The vision in the left eye was 6/6--.

I shall be interested to know of any further development of your luminous stimuli.

Very sincerely yours,

(Signed) HAZEL A. WENTWORTH."

It is believed that the foregoing will encourage and strengthen the efforts of those in our profession already believing in and using colour field interpretations; and further, that it will act to encourage others to enter this field of exceptional interest; however it is not desired to urge the profession on into an endeavor that is only a fantasy, but rather that, as a thinking profession, we should expect to find a foundation for the results that are offered so that they may have more meaning. There are those, for instance, who will not accept colour fields as taken at one sitting because they do not present a picture which can be checked up with some existing text-book on the subject denoting a certain type of failure. There are still others who base all of their reasoning on the interlacing and overlapping of the various colour fields.

The writer, however, believes that when it is shown that colour fields are, in the main, only a more delicate test than that of white (and that has been proven), a consideration is required which is very similar to that required for visual fields of white with this great difference, viz., because they are so sensitive their value lies strictly in comparison; and unless two fields are taken within the same twenty-four hours (probably five to seven hours apart) the true value will not be exhibited, so it is the changes in colour fields with which we are mostly concerned. If, for instance, we were to state that in focal infections the field for green is definitely constricted (with the amount of construction being varied by the type), and then returned to normal size upon elimination of that infection; or, if we were to say that in certain types of toxemias the field for red is contracted after the entrance into the system of a specific toxicant, and returned to normal upon its elimination; or a great amount of the reasoning which will be given in detail under the chapters so named, we, as a reasoning profession, should want to ask the question, and at the same time by research and study, answer it—"Why do colour fields change?"

A searching investigation of all the available material relative to the changes which take place in the visual interpretation of chroma (colour field studies) fails to bring to light any reasoning, designed to explain why the colour fields change. We are not considering, at this time, the types of fields as are differentiated by sex, age, or those whose differences are associated with refractive errors, but reserving those variations for later paragraphs. Let us concern ourselves now with the variations which take place during the course of a day or night, and as they may

be associated with focal infections, systemic toxemias, organic involvements, and scotomatous development—sometimes of a degenerative nature. The writer does not desire to claim a knowledge of the secret and mystical word of explanation, but rather to place before the readers some explanations of a theoretical nature which, after long consideration, seem to be indicated. The hope is expressed that the reasoning may appear to be logical, and that it may be the inspiration causing others to delve more deeply into what seems to be an absorbing and interesting research. There are probably four outstanding reasons that we shall consider. They are as follows:

1.—The chromatic fragility due to stage of development

2.—Retinal circulatory changes

3.—Retinal stretching

4.—Cortical deficiencies.

In previous chapters, we have thoroughly analyzed and stated the stages of development in connection with our colour interpretation. Thereby, it is agreed that our primitive vision was purely achromatic. In other words—that it consisted of black, white, and the black-whites, or greys. The chromatic sensations first developed were the unitary and complementary yellow and blue, followed in the third stage of development by the unitary red and green. The two latter, i.e., red and green being the most recent, are also the most fragile; and we find them first and most easily affected—in fact, they are altered quickly in primary infections and slight intoxications. In early stages of optic atrophy, they are first to be affected, and in fact, vanish with its progress. Functional derangements, even though they may be slight, will register their effect, and be evidenced by alterations in these, the smallest of the chromatic fields. Stepping back, then, to the second stage of development, i.e., yellow and blue, we find in them a combination of more hardy chromatic sensations materially affected only as the infection goes into its secondary stages, and then usually only slightly. As the aforementioned functional derangements are continued in without correction, they reach a stage when the organs become involved; and these organic involvements express themselves in contractions of the blue. Not much clinical evidence is at hand concerning the changes which take place in the interpretation of yellow, as very little testing has been done with that colour.

The achromatic sensations, which, as before stated, were the first or primitive, are also the most sturdy; and it is only after degenerative processes are undergone that they are affected. This matches very nicely with all of our previous work, in that the extreme periphery (where this achromatic sensation is found alone) is always last affected in the diseases of which we are cognizant. The mid-periphery (where we have added to the achromatic, the chromata yellow and blue) has its affections associated with the more serious of the non-degenerative changes. Lastly, the central and immediately adjacent paracentral areas (where we have added to the aforementioned, the chromata red and green) are affected quickly even

by slight amounts of fatigue, recovering just as rapidly.

Retinal circulatory changes, i.e., changes in the quantity and quality of the blood supplied to the retina, has a logical significance when we consider the plan of the circulatory system in operation. We know that as the four main arterial branches enter by way of the optic nerve head they are immediately directed into the four quadrants of the retina, viz. above, the superior nasal and the superior temporal, below, the inferior nasal and inferior temporal. Each of these branch and rebranch into the more remote areas of their individual quadrant, separating further and further as they approach the periphery, so that there is an increasingly poor supply as we leave the central area. Of course, in the branching of the superior temporal and inferior temporal, there is almost an interlacing of the two just to the temporal side of the optic disc, resulting in a very rich blood supply in the region known as the fovea centralis and between that and their point of entrance at the blind spot of Mariotte. It is known further, that different types of toxins have different effects upon the blood vessels, more noticeable, of course, in the hair-like capillaries which mark the ends of the blood vessels and their branches. Therefore, it is logical, presupposing that the colour sensations, like all other sensations, are affected by the blood, to reason that a toxin exerting a constriction will have as its result a lessening of the amount of blood supplied to the area in question; also when the effect of the toxin is that of enlarging the hair-like vessels, the area involved becomes engorged with blood. This gives us a reasonable explanation of the change in quantity of blood, and when we fit into that statement the fact that the quality is changed by the entrance of toxins, we have a basis of understanding.

Let us apply this reasoning to that aforementioned area between the fovea centralis and the optic disc; and we find that it gives an explanation for the susceptibility of the area in which we have the red and green sensation. In fact, the writer has seen fit to call this area the *Amaurotic Triangle*. This triangle has for its apex, the fovea centralis, and for its base the optic disc. De Schweinitz, in his book, "Toxic Amblyopias," spoke of finding failures in this area in the interpretation of red and green associated with various amblyopias such as tobacco, alcohol, caffeine, and other drugs. But, we wish to differentiate amblyopia (dimness of vision) from amaurosis (loss of vision), and say that we find most of the indicative scotomata, even of a degenerative nature, in this area. In fact, the changes in the size of the blind spot which have the most significance are those enlargements which take place toward the fovea centralis. The Bjerrum sign, Seidel sign, etc., usually involve this fixing area.

Now, applying this same reasoning to the mid-peripheral area where the vessels are running out and thinning out, the following might be considered as logical, viz. that where continuous functional derangements (even though they be ever so slight) result in organic involvements with which are associated sclerotic changes in these end vessels, we probably have an accounting for the fact that the interpretation of blue is thus

affected by the possible anemic condition thus created in this area. This may also be accountable for the fact that in age the field of blue becomes smaller, the amount being measured not only by the age of the individual, but also by his state of health.

Traquair, in his book on Page 232, discusses the blood supply of the visual nerve path as follows:

> "THE BLOOD SUPPLY OF THE VISUAL NERVE PATH.—Only those features which have a bearing on field defects are referred to here. The pigment cells, rods and cones, and outer nuclear layer of the retina are nourished by the choroidal vessels which are quite separate from the retinal vessels except where the two sets anastomose in the neighborhood of the optic disc. The meshes of the choroido-capillaris are especially close and regular in the macular area to which, according to Hepburn, a special arterial twig is supplied. The posterior half of the choroid is supplied by the short ciliary arteries, the anterior half by the recurrent branches of the long posterior and the anterior ciliary arteries. Posteriorly around the optic nerve entrance and at the anterior peripheral part the arterial twigs anastomose in the choroid, but the intermediate zone is practically free from anastomoses. It has been inferred from this that in certain pathological conditions the circulation in the intermediate zone is more liable to failure than that in the anterior or posterior portion, a view which forms the basis of one of the suggested explanations of the ring scotoma in retinits pigmentosa."

Looking at the retina now from the standpoint of depth, which is the consideration of the foregoing quotation, and bearing in mind that Traquair points out an anastomosis existing in the area described before as the *Amaurotic Triangle,* between the choroidal vessels and the retinal vessels, we have a further explanation of the susceptibility of that area to scotomatic changes.

In a previous chapter, we discussed the arrangement of the rods and cones in the retina, and associated with that, the fact that colour recognition varied as to arrangement of the rods and cones. Microscopically, we have ascertained that at the fovea centralis, we have nothing but cones; at the ora serrata, or periphery, we have nothing but rods, and that the cones are far less in number than the rods; so that in considering the various retinal areas from the fovea centralis in the direction of the ora serrata, we find the cones rapidly replaced and supplanted by rods—the theory, of course, being that in the animal (early state), retina, we have nothing but rods, which took on the higher stage with the evolution of the human, increasing in their development toward the fovea centralis, or area of fixation. This, of course, to allow the human the best visual apparatus for its needs.

In reiterating the areas of colour sensitivity in the retina, we find that at the fovea centralis we have all colours recognized normally. At the ora serrata, no colours are recognized (it being the area of purely achromatic vision). In moving out toward the periphery, as stated before, we first drop off the red and green, then blue and yellow—finally having nothing remaining but the black-whites, or greys; so that if one should

take any area of the retina, he would find the proportion and relation of rods and cones to vary according to the area observed. The closer to the central area, for instance, the higher the percentage of cones; in the periphery, a very high percentage of rods—in fact, very few cones, if any—so that there is an association between the colour sensitivity of the area and its proportion of rods and cones. Logically, then, if we remember that colour sensations are transmitted by the cones, it is reasonable to expect that if anything is done which further separates those cones, a resulting reduction in colour sensitivity will be had. This is probably a good explanation for the fact that in myopia where there is usually an axial extension, the fields are consequently smaller, and it must not be considered in the light of a great extension being necessary, because the factors under consideration, viz. the rods and cones, are so small that any small variation would immediately be effective. This is probably also the reason for the fact that glaucomatous changes (as has been before stated and proved) are first observed in colour before indicative information can be obtained in the field of white.

We find an interesting corroboration of the two reasonings last given, viz. retinal circulatory changes and retinal stretching, in Traquair's book, on Page 73, in which he sees fit to combine the anatomical or physical with the physiological, as follows:

> "As regards the photochemical apparatus, alterations in nutrition are brought about in several ways. Inflammatory foci in the choroid or defects in the choroidal or retinal circulation may lead to oedema, or to cicatricial or degenerative changes in the retinal outer layers. The rods and cones may become loosened from the pigment epithelium as in the initial stage of detachment from whatever cause. A general lowering of nutrition, or vitamin deficiency, as in war hemeralopia or xerosis conjunctivae with hemeralopia, can apparently affect the retinal outer layers, though here the difficult region of what is called functional disorder is approached. To what extent toxins act directly upon the rods and cones is unknown; the night-blindness of jaundice is probably due rather to the absence of fat-soluble vitamin than to the presence of toxic substances. There is evidence also that damage or functional impairment may result from an over-exercise of the normal metabolic processes such as is induced by excessive light. Direct damage may be inflicted by tumour growth, but the visual results of this injury on the outer layers have not been studied. Lastly, primary degeneration may occur, the choroidal circulation being apparently intact."

Cortical deficiencies—that is to say, deficiencies in the receptive and perceptive cortical areas of the brain—are indeed a very large consideration—one in which we will probably be foundered, so that only a brief consideration will be allowed us here.

The visual neurons are without a doubt very sensitive to nutritional alterations so that impairments and vitiations by toxic agents will express themselves through the activities of the nerves. There are many ways in which the arterial supply may be constricted or the venous circulation be congested, but either or both can be produced by the entrance of toxins into the lymph spaces, and by that path into the blood. Among the other ways may be included traumatism, mechanical pressure, injury, and

cicatrisation. Traquair mentions:

> "*Vide*, the well-known physiological experiments in which conduction is blocked by exposure of a section of a nerve to alcohol vapour, CO_2, oxygen deprivation, etc."

In fact, the visual nerve path is so sensitive to changes that it is by virtue of this that visual testing (in other words, Optometric procedure) requires such careful Technique; and further, the findings thereof are so indicative as to be of extreme value even in general diagnosis. The neurologist, therefore, is very interested in visual changes, particularly those of colour.

Although the nerve fibers are characterized by a high power resistance, they are first to be affected by impaired nutrition, and this defective conduction is associated with loss of interpretation of the red and green. This has a very great significance in that if the fields are proportionately altered, the condition may be considered as either of long standing or having a degree of permanency. To the reverse, with the fields altered disproportionately (that is to say, with the blue maintaining approximate normality, and the red and green showing alterations), the condition causing this may be considered as active and susceptible to quick progression or regression. At the present time, it is not possible for us to associate—as it would seem some of the users would require for their acceptance—certain field changes with certain diseases so that the lazy and inefficient practitioner might have a "rule of thumb" method which would spell for him the secret word of success, but rather do colour fields at this time require the thinking, intelligent interpretation of a profession based on scientific foundations. What the future will hold is a matter of interest which only experimentation and clinical data will determine.

Then, it is probably due to the fact that receptive and perceptive centers reside in the brain where the entire mechanism of direction seems to take place, that we have the psychic changes expressed. For instance, it has been demonstrated that blood pressure can suffer a lowering of from 20 to 50 points during sleep as compared with that during activity. In fact, blood pressure is usually higher at the immediate moment of first taking than it is a few minutes later when the patient is more composed. If this be true, we have a ready explanation of some of the difficulties experienced by technicians in colour field charting when they claim that the size of the field will vary somewhat in a few moments. This is to be expected, and has a logical basis for its explanation. In fact, in hysteria, which is an advanced step in the same direction, we have sometimes a complete loss of colour fields, usually in a rapidly decreasing spiral; and upon the patient being quieted, just as rapid an increasing spiral shows their return to probable normality. This is very likely also the reason why we can expect to find the colour fields of the female more susceptible to change than the male. In fact, several of the forms of psycho-neurosis are more prevalent among women than men, because they are more emotional and more easily upset, depending on their emotions to win for them that which they would fail to win by reason or force.

Dorsey has said:

> "There is no sharp distinction between psychoneuroses and dementia—a form of insanity—and they may be due to faulty emotional habits, sex complications or moral conflicts, selfishness, love of self or some unattainable object, lust, envy, jealousy, fatigue, organic diseases, emotional storms, and disorders of personal adaptation masquerading as physical ailments."

Although the writer is fully aware of the probability of weak spots in that which has just been stated, he is further aware of the impossibility of anyone giving or even knowing the real explanation of the fact that colour fields do change during a short period. Therefore, that which is presented here is not free from objection, but is nevertheless offered as an expression designed to place this great Optometric possibility under the spotlight of attention. It is felt that a consideration of these causes (even though they be theoretical) is the ground upon which we must find the diagnostic value, and a great part of the remaining chapters of this treatise will be devoted to an attempt to list in understanding fashion the diagnostic indications that are available at this time. That the future will enhance this knowledge is our belief, and we hope to be compelled to greatly enlarge on the material presented in the light of new information. Therefore, until a "rule of thumb" can be accurately worked out (and we do not believe that it will ever come about), we must look upon our chromagraphic work, bearing in mind when we study a set of fields as compared to another, that the following named factors will be contributory evidence, altering and explaining that which we have under consideration:

1st—Slightly larger fields can be expected in hyperopia; slightly smaller ones in myopia, so that any so-called normal field which we may set up may vary in the neighborhood of five degrees by virtue of the refractive error of the subject under examination. Perhaps some day (as has been stated before), we may be able to take the dioptric figure representing the refractive error and use it as a multiplier or divisor of certain so-called "normal" figures, thereby clarifying their apparent failure to meet set calculations.

2nd—Although age up to forty years does not seem to be a great factor, yet we can most certainly expect to find slightly larger fields in youth and slightly smaller fields in age. Here again some day we may be able to assist the helpless by using the patient's age as a factor mathematically, altering and thereby explaining the variation of a set of fields from a so-called normal.

3rd—The factor of sex, although not entering into a changing of the size of fields usually, it most certainly is a factor responsible for the amount of variance and the speed of the changes exhibited in a daily comparison; so that if we bear these three factors in mind, having used a Technique which will allow for the greatest amount of standardization in the mechanical and physical factors involved in taking fields, we ought to be able to better understand the changes which will be hereafter set down.

HENRY THE EIGHTH*
Some Medical and Psychological Speculations Concerning His Late Majesty's Health

Dr. Earl J. Deane
Bloomsburg, Pa.

Whether "great men" have had any real influence on the world, or whether history is merely a matter of human ideas and tendencies, are questions highly debatable. To what extent the actions of great men were influenced by their health is a highly interesting field of Speculation. We suspect that the mental and physical conditions of many were abnormal when they were driven to take actions which have passed into history.

However, while we know too little about many to dogmatize, there were many whose actions were clearly the result of their state of health. One of these, Henry the Eighth of England, was a leader during a critical epoch and we venture to study the cause of his actions from the medical viewpoint—a viewpoint much neglected by historians.

While our background may appear to be unsentimental and harsh, in medical opinion, it is probably true. If our attitude appears iconoclastic we are sorry, and can only wish that the light we cast upon the subject of our study were more sentimental and beautiful. The fact that Henry VIII was subject to unhappy human frailties in no way detracts from his renown.

In an effort to medically interpret the actions of King Henry, we will find no helpful facts in the much Bowdlerized sketches that most of us have learned at school. The great mass of available material concerning Henry and his time is shot through with theological and ecclesiastical bias. In the eyes of many Protestants Henry was a saint, while many Catholics believe that, if he was not the devil incarnate, he at

*Editor's Note:—

This paper was prepared by Dr. Deane while assisting a group of college preparatory students who were making a survey of Henry VIII and his time. Obviously, had Henry VIII's health been better England's history might have been very much different.

To quote Dr. Deane, "it was prepared with no thought of muck-raking or scandal mongering. I have simply exercised the doctor's privilege of speaking quite plainly about a very serious matter."

While the presentation of the subject is perhaps unique, we believe it will be of interest because probably fifty per cent, or more, of the cases of iritis seen by the ocular practitioner are of luetic origin.—C. C. K.

*Submitted for publication February 21, 1930.

least had the cloven hoof, horns and a tail. Both opinions are held by many present day people who ought to know better. The facts are otherwise. To obtain a clear medical perspective of Henry's actions, we must first divorce him from the position erroneously given him in relation to the church. At this late day one more divorce will surely not annoy him.

It is commonly taught that Henry VIII of England was inspired by his "lust," and attempted Papal control of his appetites, to turn on the Pope and question his power in England. This superficial view which represents the Reformation in England as a mere expression of Henry's perversity is an insult to the intelligence of scholars. Long before Henry's time nearly one-fifth of England had embraced the more radical teachings of Wyclif. All Europe was ready for a reform, and was prevented from embracing it only by the smell of gunpowder. Long before the question of Henry's divorce arose, many of the learned clergy were preaching a radical protestantism and had many followers —in fact the Dean of St. Paul's was a leader of the movement.

Regardless of what we may think of the character of the man who displaced the Pope as the head of the Church of England, any student can easily see how he could reach his conclusions in sincerity.

English kings had long refused to recognize Papal supremacy in the form demanded by the Popes, and royal advisers like Cranmer, Archbishop of Canterbury, had studied Lutheran ideas at Cambridge and in Germany. To consider Henry's lusts and ambitions as the cause of the English Reformation is, to the real student of history, amusing. The contrast between Christ's teaching and the teaching and practice of Rome at that time was so great that, no matter what "occasioned" a man to reflect on it, he could hardly be other than sincere in saying that he perceived it.

Having set the stage we proceed with the facts of our medical survey. Many facts will of course be omitted, as we do not wish this paper to be a mere "chronique scandaleuse." At the outset we present a quotation from Sir William Osler, "Know syphilis in all its manifestations and relations, and all other things clinical shall be added unto you."

Whether syphilis was known in Europe prior to 1493 is still a subject of debate. Some authorities insist that there is no evidence of pre-Columbian syphilis before the return of the Spanish sailors from Haiti, from whom it spread among the inhabitants of Barcelona. It reached Italy in 1493 with the army of Charles VIII. The disease spread through Italy, and in a few years all Europe was aflame. Some writers contend for the antiquity of the disease in Europe and Asia, relying on old Chinese records. The majority of evidence, however, is in favor of the American origin.

Syphilis (pox, lues) is a specific disease of slow evolution caused by a parasite, the treponema pallidum, propogated by inoculation (acquired syphilis), or transmission through the mother (congenital syphilis).

It is chronic in course, indefinite in duration, essentially intermittent in character, manifesting itself by a succession of lesions which may involve any part of the body. It is as varied in character as it is widespread in distribution. *No country in the world, no organ in the body is exempt from its taint.* It shortens more lives than we can estimate, and its ultimate ravages are not yet known.

Every feature of acquired syphilis may be seen in the congenital form. The intra-uterine conditions leading to the death or infection of the fetus do not here concern us. The child may be born healthy looking, or with well marked evidences of the disease. Many freakish things may happen. Of twins, one may be born healthy, the other syphilitic. A syphilitic father may beget a syphilitic child without apparently infecting the mother; yet this mother cannot be infected with syphilis by nursing the child (Colle's law), although the child will promptly infect any other wet nurse. Moreover, the mother of a syphilitic child, although remaining healthy for years, almost invariably ultimately breaks out with tertiary syphilis. Manifestly, therefore, the mother of a syphilitic child, even though she remain apparently sound, is syphilitic. So, too, a child showing no taint, but born of a woman suffering with syphilis, may with impunity be suckled by its mother (Profeta's law).

It is believed that the danger of transmission from father to child ceases in about five years if the father is properly treated. Paternal virulence may last indefinitely in untreated cases. The danger of transmission from mother to child does not, however, terminate at any definite time. Some mothers continue to bear syphilitic children for years after the disappearance of their own symptoms.

As to the child itself. It may die "in utero" and be expelled as a disorganized fetid mass. It may be born to live but a few days. It may reach maturity, bearing in mind and body the scars of its parents misfortune. It may remain well for years only to develop an unsuspected hereditary syphilis. It may not be infected. The possibilities are legion.

When a man in the infectious stage of syphilis marries, the first product of conception usually dies "in utero," and miscarriage of a deformed, macerated fetus ensues. After one, or more, such mishaps a child is born either actively syphilitic or soon showing evidence of the disease. Such children usually die promptly. Later children are born which show no signs of the disease at first, and may remain well or show stigmata of syphilitic heredity, or become actively syphilitic.

The general prognosis of syphilis is a gloomy prospect compounded of various follies amid which madness for drink and carelessness in treatment stand pre-eminent. In syphilis, as in any other disease, the most important element of prognosis is the way the patient "happens" to react to the infection. Syphilis brings out every latent weakness. It plunges the hysterical and the neurasthene into new depths of despair. It feeds every organic neuropathic predisposition. In short, syphilis

assumes the habit of its victim. Its course depicts his weakness. Syphilis, like character, is founded upon heredity modified by environment. Every influence to which the patient is subjected may react upon his disease. Time and treatment are the enemies of syphilis. Age certainly does not wither her though time cannot be said to stale her infinite variety.

All efforts to develop artifically either an active or a passive immunity to syphilis have failed. The results of experiments have been absolute zero: as yet *"the only way to be immune to syphilis is to have it."*

Many of Henry VIII's actions were undoubtedly results of the syphilis that eventually killed him. The records tells us that he died in a coma, or stupor, probably uremic—the usual termination of the complicating cardio-vascular-renal disease that always accompanies an untreated and neglected syphilis.

Having made a diagnosis, we proceed to support it with the results of Henry's marriage record. Having said that Henry had syphilis the question, "When and where did he get it?" will probably be asked. According to the record he does not seem to have obviously infected any of his wives. He was probably infected several years before he married Catherine of Aragon. Even though he was barely nineteen when he married her, when we consider the court life of his day, a gay young prince like Henry had a multitude of opportunities to catch it any time between the ages of fourteen and eighteen.

It is probable that his primary lesion, so apparently innocent and harmless, healed up under the influence of some simple ointment (Dr. Welsh points out that many ointments in use at that time contained mercury), just as it does today in many men, who will bitterly rue the apparently trifling incident that later on causes them much woe. It is the cases of syphilis that appear trifling at first which, under the influence of worry, lack of treatment, overstrain, etc., that go tragically at the end. There is a wicked little pun that is common among medical men. "Three minutes with Venus may mean three years with Mercury." Many people believe that the only nervous results of syphilis are partial or total paralysis. Among neurotic people it may cause serious mental trouble without apparent damage to the brain tissue. In the Oxford Textbook of Medicine we find this statement, "Such patients are generally psychathenic, ill balanced and degenerate." The writer is speaking of the mental effects of syphilis and its propensity to cause "phobias" and "obsessions."

The idea that Henry suffered from syphilis is reasonably explained by the following facts—

(a) The extraordinary number of premature births and dead children from two of his wives, one of whom was young and apparently healthy.

(b) The poor health of at least three of his children, Mary Tudor, Edward VI, and the Duke of Richmond. After a look at Elizabeth's health record one doubts that she escaped the infection.

(c) The terrible degeneration—mental, moral and physical—that

set in in early middle age.

(d) From an able and athletic man he became a mass of loathsome infirmities. So bloated and unwieldy that he could hardly pass through a door—his legs swollen and covered with festering sores, causing an unbearable stench.

(e) The sinus in his leg which caused him unbearable agony whenever it was closed. It was probably a syphilitic periostitis occurring in an essentially neurotic man.

(f) Death in stupor at the comparatively early age of fifty-five.

The number of premature births endured by his wives is in dispute. All sorts of stories are told to show that his children, if born alive, or his wives if they became ill were especially subject to the effects of cold and overlong christenings. *Once* would be all very well: but when it happens more than once the doctor becomes suspicious. One has but to read the record of the treatment of his wives to better trace his great degeneration. Let us now look at a short summary of Henry's marriage record.

Of his first wife, Catherine of Aragon, we have the following record. On January 31st, 1510, seven months after her marriage, she gave birth to a daughter still-born. Eleven months later, on January 1st, 1511, there was a son who died in three days. In September, 1513, there was another son, either still-born or dying immediately after birth. In June, 1514, there was another son, no sooner christened than dead. Then on February 18th, 1516, came the little princess who became "Bloody Mary." Then in 1517 the record says, "it is probable that there were several miscarriages." On November 18th, 1518, came a boy and still-born. Poor Catherine had done her best but her husband's syphilis was too much for her. That she undoubtedly died from a syphilis gained from her husband is shown by the account of the post-mortem examination of her body. The report says that she was all sound but the heart, which was "black and hideous, with a black excrescence which clung closely to the outside." No doubt an aortic aneurysm, a common result of untreated syphilis.

The first appearance of the frightful physical degeneration of Henry was during Anne Boleyn's time. It was probably from that circumstance that Anne was accused of being the prime mover in his degeneration. It seems unfair of Yorke, who in his sketch of Anne Boleyn said, "she appealed only to Henry's lower nature." Some other woman had effectually done so, and years later Anne had to suffer for it.

Granting that Anne was a free-spoken and somewhat vindictive woman, it is hard to believe that the industrious, dancing and singing little queen was wholly to blame for the alleged acts that brought her pretty head to the block. She was probably a victim of nymphomania, or, more properly, puerperal insanity—a pathological condition of the nervous system that does not necessarily imply that a woman is of abandoned character. After the birth of Elizabeth, Anne had three mis-

carriages. Again these premature births! Surely they were due to Henry's syphilis!

Henry next married Jane Seymour. In about a year she was delivered of a son, who survived the perils of infancy only to die of what was probably pulmonary tuberculosis.

Next came Anne of Cleves, the famous "Flanders Mare," whom Henry promtply divorced on the plea that she was not "virgo intacta." She was followed by Lady Catherine Howard and, in January, 1542, Parliament relieving the blubbering and neurotic Henry of responsibility, set the stage for her beheading in the tower.

It was during Catherine Howard's time that Henry's greatest degeneracy was coming on and his mental syphilis was approximating to its greatest terrors with all its obsessions and phobias. The ulcerated sinus on his leg sometimes closed, and the pain was so great that he became speechless from agony and went black in the face. How neurotic he was at this time! Daily did he grow more corpulent and the ulcerated leg become more offensive. When he went to the north he cleared the tower by beheading every prisoner in it. Such actions were undoubtedly symptoms of a syphilitic psychasthenia, of a terrible physical and moral degeneration. If not, what are they? Symptoms of mumps, perhaps.

Next and last came Catherine Parr. She must have been a brave woman to take on the task of nursing Henry, whose temper by that time was like a wild beast's because of obsessions and phobias.

So stands the record! A record of disease, lust, obscenity, grandiose ideas and violence combined with cowardice. All very typical of syphilis. It is impossible to say how many lost their lives because of Henry's syphilitic obsessions and phobias. It is quite possible that when Sir Thomas Moore said, "Her Majesty Queen Anne Boleyn may dance and sing but her turn may shortly come," that his acute mind saw that his King was not mentally well.

Whatever else we may say of Henry we cannot say, "psycho-analytically," that he suffered from "repression of the sex complex." On the whole, one cannot avoid the conclusion that his conduct to his wives was not all his fault. They seem to have done no credit to his power of selection. Considered as women, the first and the last appear to have been the best.

In syphilis and self-indulgence we have the secret of the tragic development of Henry's character. He abandoned all moral restraint and his degeneration was the result of syphilis, gluttony and bibulous self-indulgence.

As in Henry's day, so too, today does syphilis continue to destroy the flower of the human race. Hard-working, intellectual, middle-aged men who once upon a time were very human youths. Altogether too many of our youths "go to the big county fair" wholly uninstructed, or inadequately instructed, as to the dangers and ravages of syphilis. It is a problem that should have the serious attention of parents, teachers,

clergy and doctors. In conclusion, we have drawn freely and without stint from our bibliography; whole paragraphs have been quoted verbatim. Time and space forbidding detailed references, this brief acknowledgment stands as an inadequate voucher for a very heavy debt.

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OPTOMETRIC CASES TREATED WITH PHYSICAL THERAPY

The optometric application of ocular physical therapy is, to be sure, still in the early stages of its development. Despite this fact, however, we have received a number of case reports which illustrate the advantages of using this type of therapy in addition to other optometric treatments. Today optometrists in all parts of the country are successfully using electro-therapy in their practices and the following cases are given to illustrate the success they are having:

Case of Squint.

Patient—Mr. G. P.—came to my office 4/24/29—Occupation, electrician—Age 29 years. History: Claims poor vision in left eye since 1915; gets headaches, continual blinking, left eye turned in for five years.

Ophthalmoscope—O. D. Normal; O. S. Normal.

Esotropia 15Δ but hard to get both eyes to work at the same time; has never worn glasses before. Rx: given +100 D. S.—O. U.—V. O. D. 20/20; V. O. S. 20/40.

I treated this case with Diathermy three times a week for twenty to thirty minutes, massaged eyes, temples and back of neck with high frequency current, and also prescribed home exercises.

At present the patient's vision is O. D. 20/20; O. S. 20/30+1, and eyes are straight, headaches and blinking ceased.

Case of Exotrophia.

Mr. W. D., Gary, Indiana, February 9, 1927—Nationality, Polish. History: Claims left eye turned out eight months ago; unable to move;

has been advised operation; has had no results from glasses. Vision O. U. 20/20, but cannot use both eyes at same time unless turning his head to one side.

Rx required but not prescribed: O. D.+75 D. S.; O. S.+50 D. S. +25 D. C. Axis 180. Exotrophia 45Δ in left eye; treated exclusively with diathermy with one electrod on 4th, 5th, and 6th cervical and other electrod over left eye for 12 to 15 minutes, using prisms only to determine imbalance. I did relax eye with high frequency for 5 to 7 minutes; treatments daily for first two weeks, then thrice a week, cutting treatments down gradually to once a week; discharging patient May 4th, 1927, with add 40Δ and 2Δ esophoria.

Case of Muscular Imbalance.

Miss J. M., Gary, Indiana—Age 17 years. Claims has headache over eyes for some time. Vision: O. D. 20/40; O. S. 20/40 Exo. 7Δ L. H. 4Δ eye would turn up at times and attention had to be given as she would pull it down. Gave Rx O. D.—50 D. S.—75 D. C. Axis 180; O. S.—1.00 D. C. Axis 180; Vision 20/20 O. U.

Gave diathermy daily over both eyes and sinusoidal (slow), reducing treatments as case progressed, discharging patient in three months.

Dr. H. R. Spitler,[2] who is also carrying on considerable research along this same line, reports two cases (the writer will quote only one) of senile cataract treated successfully with the application of spectroband.

A Case of Cataract.

Case 1. Mrs. B. Female, anabolic, aged 64. Right eye fully developed cataract, left eye incipient with vision only 5/200. Light perception and projection with each eye. Prescription: 24 to both eyes for fifteen minutes daily for ten days and alternate days for fifteen more irradiations. Systemic 23A alternate days. Results—vision left eye was 20/100 with ability to count fingers held before the right eye at a distance of four or five inches. Patient discontinued treatment and was not seen for seven months, during which time a trade marked lens was used as a goggle, at which time vision in the left eye was 20/70, with no noted change in right eye. In view of the fact that this patient did not take the full number of treatments prescribed, the results seem truly remarkable.

Dr. Cox[3] also reports several cases which have responded to various forms of physio-therapy application, particularly with reference to the treatment of senile cataract.

Two Cases of Cataract.

Mrs. L., age 71, health good. Right eye media clear and vision 20/20 with plus 3.00 lens. Left eye amblyopic; light perception only. Absolutely no vision of forms, similar plus 3.00 bifocals with 2.50 adds were ordered. Reported within one month some vision of forms with left eye. Came again June, 1926, complaining of fogginess and could not read. Ophthalmoscopic examination showed a very cloudy lens in right eye. Five diathermy treatments of which the first was only 25 minutes were given, the remaining four were 45 minutes diathermy and four minutes

surging sinusoidal with five volt strength. The right eye completely cleared with full 20/20 vision and has remained so to July, 1928. Left eye showed decided improvement in vision and has continued to improve until she could read letters one-fourth of an inch high.

Wm. C., age 70, good physical health. Cataracts in left eye; light perception only. Right eye, through a very small opening on temporal side could just make out big E at two feet. Vision improved with treatments three times a week. Vision after fifty-second treatment 20/50, enabling him to carry on his farm work.

The writer also,[4] has had no little success in the application of physiotherapy in certain cases. The following case is, I believe, a splendid example of the value of diathermia in amblyopic treatment work.

Case of Amblyopia.

Mrs. T., age 52, called for a refraction June 13, 1929. She claimed that the left eye had been blind for several years and that she has been losing the sight of the right eye for the last few months. She visited several ophthalmologists but was told nothing could be done to save the sight of the right eye. The ophthalmoscopic examination revealed atrophy of the left optic nerve. The fundus of the right eye was apparently normal, with a slight cupping of the disc. Static skiametry O. D.—6.00 D. Sph. ⌒ —3.00 D. Cyl. Axis 30. O. S. —9.00 D. Sph. Dynamic Skiametry O. D. —3.50 D. Sph. ⌒ —3.00 Cyl. Axis 30. O. S. —7.00 D. Sph. Vision without lenses O. D. Fingers at 8 ft. and O. S. light perception only. Vision with static skiametric findings was O. D. 20/200; O. S. light perception only. The correction worn for the last 12 years was O. D. —4.00 Sph. ⌒ —4.00 D. Cyl. Axis 180 V. = 10/200; O. S. —8.00 D. Sph. which did not improve vision. The case was diagnosed as a case of amblyopia in the right eye and amplyopic treatments were instituted. A combination of physical agents consisting mostly of high frequency and diathermy of varying techniques were used, the treatments were given twice daily, for a period of one month. The following lenses were prescribed: O. D. — 4.50 D. Sph. ⌒ — 4.50 D. Cyl. Axis 30; O. S. — 9.00 O. Sph. During the first two weeks there was little improvement in her vision, after this period improvement was rather rapid. The notes on our records are as follows: July 1, 1929, with above correction O. D. V = 20/100 O. S. no change during the entire treatment period; July 5th, O. D. V = 20/70; July 9th, O. D. V = 20/50; July 13th, O. D. V = 20/40. The patient being a farmer's wife living a few hundred miles from Minneapolis had to return to the farm at this time, although she expressed the desire to continue the treatments for a longer period and she promised to return for treatment within the next three months as soon as the farm work would be completed. On October 21, 1929, the patient again reported at the office claiming that the sight is not nearly as good as at the time the treatments were discontinued. Upon examination it was found that the acuity of vision in the right eye had dropped to 20/100 during the three months she was away. A search for the cause of the reduction in visual acuity had been made as I suspected some foci of

infection and while the patient claimed to be strong and healthy and looked the part, she was sent to a reliable physician for a complete medical examination. The report of the examination was that there are some definite foci of infection present and the patient was advised to have that condition attended to. Treatments of ocular physical agents were again instituted and the sight was gradually improving. Her vision in the right eye was 20/25 on December 12, 1929. On account of sudden sickness in the family, treatments were again discontinued at this time but may be resumed later.

These are only a few of the many cases successfully treated by optometrists who are applying Ocular Physical Therapy along with their other optometric techniques. Of course this, like any other form of therapy, will fail in some cases but of the number of cases treated so far, it seems that the percentage of success is large enough to encourage us all to carry on our researches in this promising and as yet but little developed optometric field. J. I. K.

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BOOK NOTICES

THE PRINCIPLES AND PRACTICE OF OCULAR PHYSICAL THERAPY FOR OPTOMETRISTS. By J. I. Kurtz, B.Sc., A.O., F.A.A.O. Limited Edition. Published by the American Journal of Optometry. 250 pages, 51 illustrations. Cloth $4.50. 1930.

The Principles and Practice of Ocular Physical Therapy for Optometrists has been written for the purpose of introducing this interesting subject to the profession. The book is divided into four parts, the first portion of which deals with the Physics and Biophysics of Ocular Physical Therapy. The second part deals with the practical clinical application of electro-therapy, describing the different techniques and when to apply each technique in specific optometric cases such as asthenopia, amblyopia, cataract, headaches, muscular abnormalities, myopia, etc. Part three is devoted to Light Therapy and Psycho-Therapy. Here the author discusses the subject of light and the effect of "Light upon Life," giving also the therapeutic effects of both the visible and invisible portions of the spectrum. He also discusses the effects of the mind upon the body, contending that mental disturbances may be the cause of muscular and nervous ocular abnormalities, also claiming that some of the most difficult cases may be materially aided by the proper use of "Applied Psychology." Several case reports are given and he illustrates how this may be properly applied by the optometrist.

Part four deals with Mechano-Therapy describing several methods for the mechanical treatment of strabismus. Special attention is given to the opto-spoon technique as well as to the finger treatment method. In this connection it might be said that there has been nothing published prior to this time on these mechano-therapy techniques in any of our optometric literature; this material being original with Dr. R. W. Bennett who has written this chapter for the author. Two other chapters were also contributed, one on the "Consideration of the Energy Transformation in the Body from an Electrical Standpoint," by J. O. Perrine, Ph.D., Electrical Engineer of the Department of Development and Research of the American Telephone and Telegraph Company, New York City, and the other on "Galvanism for Optometrists," by Dr. H. Riley Spitler.

The book is profusely illustrated and covers the field of Ocular Physical Therapy as thoroughly as the present day knowledge of this subject will permit. The author has had the student as well as the practitioner in mind in writing this text and this work is, as far as known, the first and only optometric book written on the subject of ocular physical therapy.

The first edition has been limited in quantity and by personally controlling the distribution of the book the author hopes to place the knowledge of this new subject in the hands of the progressive professional optometrists only, who will, it is hoped, find it an advantage to add Ocular Physical Therapy to their treatment technics.

L. L. K.

A TRANSCRIPT OF THE POST GRADUATE LECTURES OF THE OPTOMETRICAL SOCIETY OF THE CITY OF NEW YORK AND THE BROOKLYN OPTOMETRICAL SOCIETY. Edited by J. Robinson Cohen, Opt. D., and Wm. Feinbloom, Opt. D. Published by the Optometrical Society of the City of New York, D. K. Uttal, Opt. D., Secretary. 2482 Broadway, New York, N. Y. 112 pages. Cloth. $5.00. 1930.

This book consists of an abridgement of a series of graduate lectures given jointly before the Optometrical Society of the City of New York and a Brooklyn Optometric Society. The material has been nicely edited by Drs. Feinbloom and Cohen and the work distributed to all optometrists attending this series of lectures.

The book itself contains besides a Foreword by the Editors, six lectures as presented by various specialists and also a resumé of the work presented to the New York group by Dr. A. M. Skefington at one of his graduate clinics.

The first lecture by Dr. Wm. Feinbloom deals with the need for taking a case history, as well as the case history itself. This lecture is splendidly arranged. The second lecture by Dr. James W. Smith deals with Ophthalmoscopy. This author describes what he terms "remote" and "close" ophthalmoscopy and also the appearance of various pathologic conditions. The third portion of the book deals with objective tests by Dr. S. H.

Continued on page 523

ANNOTATIONS AND SOCIETY PROCEEDINGS

The American Journal of Optometry is the Official Organ of the American Academy of Optometry, as well as the Organ of the Arkansas, Delaware, Indiana, Iowa, Maine, Maryland, Michigan, Minnesota, Nebraska, North Dakota, Oklahoma, Pennsylvania, South Dakota, Vermont and West Virginia Optometric Associations.

The following gentlemen have consented to supply items of interest from their respective sections, acting in the capacity of

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NORTH DAKOTA STATE CONVENTION

The annual meeting of the North Dakota State Association of Optometrists was held at Bismarck on the 5th of August. Dr. Herbert N. Smith, President of the South Dakota Optometric Association, was the chief speaker. The officers as elected to serve for the 1930-1931 year are as follows: Dr. Charles P. Elliott, Valley City, president; Dr. W. H. Monahan, Williston, first vice-president; Dr. William Henning, Fargo, second vice-president; Dr. H. J. Wagner, Bismarck, secretary, and Dr. Andrew Lee, Carrington, treasurer. The next meeting will be held jointly with the South Dakota Association at Aberdeen.

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MAINE ASSOCIATION

Dr. A. L. Corriveau, Secretary of the Maine Association of Optometrists reports that the next meeting of that society will be held at Houlton on September 8th.

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VERMONT OPTOMETRY BOARD

Dr. Laurence P. Folsom, Secretary of the State Board of Examiners of Optometry for the State of Vermont announces the following new rulings for this state board. First, that all price advertising would be considered misleading and therefore untruthful, and second, that all optometrists must call themselves "optometrists" and not "eye-specialists." Failure to comply with either ruling will mean forfeiture of license to practice.

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A TIMELY SUGGESTION FROM MICHIGAN

The following suggestion, taken from the editorial section of *The Michigan Optometrist,* is one which every practitioner should study carefully. It is an important matter and much care should be exercised in laying the proper ground-work for this important step, which already has been delayed far too long.

"It must be admitted that Optometric Legislation was obtained through heroic efforts of those untiring workers of twenty years ago. Medical opposition was encountered at every angle. Through fear of defeat every Optometry law exempted the medical man. Was it intended to exempt all those who were to come in the future years? We hardly believe that was the intent of the law. It was an error once committed and then continued in every state. Can the error be rectified? We believe it can. There never has been, is not now any justifiable reason why the future Optometrists must be compelled to meet certain requirements based upon two or four years of special training and then allowing any medical man to attempt the same work with four to six weeks instruction. Which state will have the credit of first rectifying this condition? Why not Michigan? We believe the support of every first class Ophthalmologist would be extended in such an effort. Provisions could be made for the exemption of those medics who show credit from any Medical school in Ophthalmology. That would clear the field for a better understanding between Optometry and Ophthalmology."

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NEW TRANSILLUMINATOR BULBS

Two new transilluminator lamp bulbs have recently been added to the line of diagnostic instruments produced by the Bausch & Lomb Optical Company, Rochester, N. Y. These are of two types—the frontal and the dental—and each serves as an aid for the localization of focal infections and the examination of the entire oral cavity, various air sinuses and the mastoid cells. The name of these new bulbs is "Whitelite."

Continued from page 521

Roberts. Ophthalmometry, static and dynamic skiametry tests are described. The next portion of the book deals with the Phoria findings by Dr. J. R. Cohen. The author first considers the Phoria tests from the standpoint of Ocular Myology, then describes the technic of making the test itself and closes by describing the significance of the Phoria findings. Duction tests are next dealt with by Dr. C. L. Treleaven, who describes the value of the tests as well as their clinical application. The final published manuscript is one by Dr. S. S. Feinstein and is really divided into two portions, the first portion dealing with laboratory diagnostic tests such as Urinalysis and Blood examinations, including the Wassermann, and the second portion dealing with Ocular Pathology as determined in making an external ocular inspection. The book closes with the resumé of the work presented to the men by Dr. A. M. Skefington at one of his graduate clinic periods. C. C. K.

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